Midbrain meningioma causing subacute parkinsonism

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Multiple system atrophy (MSA) is an adult-onset sporadic neurodegenerative disorder characterized by any combination of parkinsonism, cerebellar ataxia, autonomic failure, and pyramidal tract signs.¹ Structural brain MRI shows atrophy of the putamen, middle cerebellar peduncle, and pons.¹ Although MSA is a rapid progressive disorder with no effective treatment, and it can present with a wider range of clinical features, several neurologic disorders underlying diverse etiologies can be clinically misdiagnosed as MSA and vice versa. Here we present a patient with subacute onset of neurologic symptoms resembling atypical parkinsonism similar to MSA due to a clival meningioma of the posterior fossae.

PRACTICAL IMPLICATIONS

Clival meningioma should be included in the differential diagnostic workup of recent onset of atypical progressive neurodegenerative parkinsonism to ensure prompt treatment.

Case report

A 67-year-old woman had a 4-month history of progressive ataxia, slight orthostatic hypotension (reduction of systolic blood pressure of 20 mm Hg after 3 minutes of standing from a previous 3-minute interval in the horizontal position), and urinary incontinence at onset. Neurologic examination showed hyperreflexia, gait ataxia, bradykinesia, rigidity, and mixed postural and resting tremor in both arms. Motor examination score on the Unified Parkinson's Disease Rating Scale (UPDRS-ME) was 51 (table). Her neuropsychological examination was normal, as was routine laboratory testing. She displayed poor response to levodopa. She fulfilled criteria for probable MSA.¹

Intriguingly, brain MRI showed a mass lesion in the left posterior fossa displacing the pons, midbrain, basilar artery, and middle cerebellar peduncle (figure, A). The mass had iso-signal intensity on T1-weighted imaging, high signal intensity on T2 fluid-attenuated inversion recovery imaging, and enhanced homogeneously with gadolinium contrast. Dopamine transporter (DAT) SPECT showed integrity of the nigrostriatal system (figure, C). The patient underwent surgery to remove the tumor via posterior fossa craniotomy. Histology confirmed a meningothelial meningioma (WHO grade I). After surgery, she displayed a slight abduction nystagmus in each eye. At clinical and imaging follow-up 2 months later, her bradykinesia and tremor became very mild and the gait ataxia improved considerably. Orthostatic hypotension slightly improved with a reduction of systolic blood pressure of 15 mm Hg. The UPDRS-ME value was 40 (table). Follow-up brain MRI showed no evidence of residual meningioma with repositioning of the brainstem (figure, B) nicely appreciated with 3D reconstruction post-surgery (figure, E). The patient gave written informed consent for research purposes but not for any filming.

Discussion

We describe a clival meningioma presenting with a picture of atypical parkinsonism similar to probable MSA with a subacute onset. Posterior cranial meningiomas constitute about 9% of all intracranial meningiomas and are classified as cerebellar convexity,

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Table Motor examination scores on the Unified Parkinson's Disease Rating Scale (UPDRS-ME)

UPDRS-ME (subsection)	Presurgery	Postsurgery
Speech	2	2
Facial expression	1	1
Rigidity	10	10
Finger tapping	4	4
Hand movements	4	4
Pronation-supination movements of hands	4	3
Leg agility	4	4
Arising from chair	1	1
Gait	1	1
Postural stability	3	1
Posture	1	1
Body bradykinesia	3	1
Postural and kinetic tremor of the hands	4	2
Rest tremor	9	5

tentorium, posterior surface of the petrous bone and clivus and foramen magnum.² Typically, meningiomas present with subtle symptoms such as headache, dizziness, vertigo,

and simple or complex partial seizures, but rarely will a gradual progression of neurologic deficits with ataxia and cranial neuropathy occur with meningiomas of the clivus.³ Meningioma is the most common tumor associated with parkinsonism and when the tumor is located in the sphenoidal ridge or the frontal or parietal areas, it is more likely to induce parkinsonian symptoms.³ Interestingly, our patient with a clival meningioma showed a combination of ataxia, parkinsonism, autonomic disorders, and pyramidal signs resembling atypical parkinsonism. The mechanism leading to parkinsonism in patients with a meningioma is not well-understood. Various reports have supposed that the pathogenesis of tumor-induced parkinsonism can be explained as a disruption in neural circuits.^{2,3} It has been suggested that the mass compresses the midbrain and interferes with nigrostriatal dopaminergic transmission, which may induce parkinsonism.^{2,3} The mass effect of the tumor can directly squeeze or deform the axon of the presynaptic dopaminergic neuron. Being the meningioma near the midbrain, it could compress the cell body of the nigral dopaminergic neuron causing the functional interruption of the synaptic integrity. Alternatively, space-occupying lesions may cause secondary parkinsonism, mainly in old age. It is possible that such lesions may mask the underlying subclinical status of parkinsonisms³; however, this latter hypothesis cannot be attributed to our case because of the integrity of DAT scan. Thus, clinicians seeing a patient with subacute symptoms suggestive of MSA should be aware of possible posterior cranial fossa meningioma. This case report highlights the importance of performing structural imaging and whenever possible an MRI in patients with movement disorders.

Figure Imaging

T1-weighted MRI shows a mass lesion (35 × 25 × 32 mm) in the left posterior fossa revealing compression and dislocation to the right of the pons, midbrain, basilar artery, middle cerebellar peduncle, and triventricular dilation, pre (A) and post (B) surgery; (C) dopamine transporter SPECT; (D) 3D reconstruction shows a 12.3 cm³ mass (red) dislocating the right middle cerebellar peduncle of 21 mm and left middle cerebellar peduncle of 23 mm dislocating the brainstem (blue); (E) diffusion tensor images presurgery and postsurgery.

Author contributions

A. Labate: study concept and design, acquisition of data, drafting the manuscript. R. Nisticò: study concept and design, acquisition of data, drafting the manuscript. A. Cherubini: acquisition of data, analysis of data. A. Quattrone: critical revision of manuscript for intellectual content.

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Disclosure

A. Labate and R. Nisticò report no disclosures. A. Cherubini serves on scientific advisory boards for Linkverse Srl and Numens Srls; is author on a patent re: Methods and apparatus for detecting and classifying skin lesions and System and method for identifying anatomical structures;

serves as a consultant for GE Healthcare; and receives research support from Italian Ministry of Health and Regione Calabria. A. Quattrone serves as an Associate Editor for *Neurological Sciences*. Full disclosure form information provided by the authors is available with the full text of this article at Neurology.org/cp.

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